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ROBERT S. DINSMORE, M.D.

Staff Member, The Cleveland Clinic Foundation, 1923-
Chief of Surgery, 1949-1956
Senior Consultant, Department of General Surgery, 1957-

Born August 10, 1892
Died September 24, 1957

ROBERT SCOTT DINSMORE

ROBERT DINSMORE died from a cerebral thrombosis in the Cleveland Clinic Hospital on September 24, 1957. The inspiration of his leadership and the warmth of his friendship will long be remembered by his associates.

In the memorial service held at the Clinic on October 5th, Doctor Dinsmore's place in American surgery was clearly described by one of his closest friends, Dr. John M. Emmett:

"Dr. Dinsmore's honesty and sincerity of purpose will live always in the memory of American surgeons of his day. He was able to make friends among all groups of people without difficulty, and he held these friends—an attribute which few people possess. One of his life's objectives was to promote the development of his younger associates. He was at his very best when he portrayed the individual attainments of one of them, and his pride in the progress of these young surgeons was apparent to all of his friends.

"Dr. Dinsmore had a splendid knowledge of the modern developments of medicine and surgery. He could scan a surgical treatise and get a clearer knowledge of the writer's objectives than anyone I have ever known. His wide personal acquaintance with American surgeons inspired him to follow with real zeal the literature which this group created. He will be remembered in our profession as a great teacher of the art of surgery, a responsibility which he fulfilled most effectively.

"Dr. Dinsmore always maintained a sufficient balance of interest in surgical science to keep him in the forefront of his profession. There has been no surgical clinician in recent years who has ascended to the heights of professional recognition to a greater degree than did Bob Dinsmore. There was no way to prevail upon him to support individuals or issues which he thought were questionable in their value. He never allowed himself to go along with a popular tide in surgery unless he felt that the issues under consideration were right. Dr. Dinsmore's counsel was sought by many and diversified groups of doctors. His answers were modulated by a wide experience and a scientific acumen which always culminated in a sense of fairness.

"Dr. Dinsmore has been described as a 'Doctor's Doctor.' I wonder if any more worthwhile tribute could be paid to a surgeon than to have his services sought by his colleagues."

THE CAUSE OF SYRINGOMYELIA AND ITS SURGICAL TREATMENT

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HYDROMYELIA is a dilatation and syringomyelia is a diverticulum of the central canal of the spinal cord. The terms are used interchangeably; the conditions frequently coexist and are clinically indistinguishable.

The Thesis

Both hydromyelia and syringomyelia originate in embryonal life as a result of distention of the central canal by ventricular fluid, caused by atresia of the outlets of the fourth ventricle. The rational treatment is to open the obstructed foramen of Magendie.

The Evidence

1. In embryonal life the central canal of the spinal cord is a portion of the closed neural tube. It becomes a vestigial structure after the foramens of the fourth ventricle open into the subarachnoid space. Conversely, if the foramens fail to open (atresia), the central canal of necessity remains a part of the ventricular system.

2. Early forms of hydromyelia and syringomyelia are present in infants having Dandy-Walker** [1] or Arnold-Chiari deformities, [4-10] while severe forms are found in adults having either of these deformities. [11-15]

3. There is evidence that both Dandy-Walker and Arnold-Chiari deformities of the hindbrain are produced by embryonal hydrocephalus due to atresia of the outlets of the fourth ventricle. [3,12]

4. In all patients with hydromyelia or syringomyelia, the foramen of Magendie is occluded. [11,12] The obstruction is due to a membrane, the attachments of which indicate that it is a persisting remnant of the embryonic rhombic roof, or to a cerebellar hernia that squeezes together the cerebellar tonsils, or to both conditions coexisting.

5. In patients having hydromyelia or syringomyelia, the ventricles usually are dilated [15] and air injected into the spinal canal frequently will fail to enter them. [11,12]

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**A bulging diverticulum of the fourth ventricle described by Dandy and Blackfan [2] in 1914 and shown by Taggart and Walker [3] in 1942 to be due to congenital atresia of the foramens of Luschka and Magendie.

6. Indigo carmine injected into the ventricles may be recovered from the syrinx.[12]

7. Fluid aspirated from the syrinx resembles the product of the choroid plexus.*[11,16]

8. In the experimental animal, inflammatory sealing off of the outlets of the fourth ventricle is followed by acute hydrocephalus and syringomyelia.[17]

9. Pantopaque or India ink injected into the dilated ventricles of the above animals passes downward into the dilated central canal of the cord.[17]

10. In the human being, distention of the central canal by cerebrospinal fluid may occur when the foramen of Magendie is obstructed by a tumor or a cyst.[11,12]

11. The symptoms of syringomyelia frequently are improved by surgically removing the posterior rim of the foramen magnum and opening the obstructed foramen of Magendie.[11,12,18,19]

The Normal Embryology

In the embryo the neural tube is a closed cavity that constitutes the anlage of the ventricular system and of the central canal of the spinal cord. The ventricular fluid leaves the neural tube by filtering through the permeable rhombic roof whence it expands the developing subarachnoid spaces.[20] The growth of the choroid plexus during the sixth to the eighth week is accompanied by a dilution of the protein content of the ventricular fluid apparently due to an increase in its rate of formation. There is an associated rapid expansion of the subarachnoid spaces and of the lumen of the neural tube during this stage.

At about the fifth month of fetal life, as the result of progressive attenuation of portions of the rhombic roof, the foramens of the fourth ventricle open. Through these openings, the pulsations transmitted to the ventricular fluid by the choroid plexus[21] are diverted from the central canal to the outer surface of the neural tube. The central canal, devoid of choroid plexus, narrows to become a vestigial structure after the development of this shunt.

The Pathologic Embryology

During the sixth to the eighth week of embryonal life, should the membranous rhombic roof not be sufficiently permeable to permit free escape of ventricular fluid (atresia), the pressure within the obstructed ventricles will rise correspondingly, causing a pressure cone of the soft embryonal hindbrain known as the *Arnold-Chiari malformation.*[12] More rarely this embryonal hydrocephalus will cause a bulging diverticulum of the hindbrain,[3] referred to

If the fluid aspirated from a cystic cord is yellow and has a high protein content, the responsible lesion is a tumor or hamartoma and not a true hydromeylia or syringomyelia.

by Benda[1] as the *Dandy-Walker syndrome*. In either case the confined pulsations of the ventricular fluid continue to be funneled toward the central canal and cause it gradually to dilate. Hydromyelia, therefore, has its onset in embryonal life, and though clinically silent in congenital hydrocephalus, it nevertheless constitutes a part of the pathologic picture. Hydromyelia often has been described[4-10] in association with the Arnold-Chiari deformity. Benda,[1] as well as Gardner, Abdullah, and McCormack,[12] has described it in association with the Dandy-Walker deformity.

The Hydrodynamics of Syringomyelia

If the intraluminal pressure ruptures the ependymal lining of the central canal, a diverticulum (syrinx) may form parallel to the canal. In an article entitled "Cervical Syringomyelia and Syringomyelia-Like States Associated with Arnold-Chiari Deformity and Platybasia," this mechanism was described by Lichtenstein[13] as follows:

> The constriction of the neuraxis by the bony ring prevents adequate communication between the caudal diverticulum of the fourth ventricle and the posterior cistern. The accumulating cerebrospinal fluid dilates the central canal and, in some instances, is associated with perforation of the ependymal lining and the spread of the fluid into the posterior columns.

The dilatation of the central canal which results in symptomatic syringomyelia obviously is a slow process and the disease seldom is diagnosed until adult life. The late onset of symptoms in patients having syringomyelia has led many investigators to consider it an acquired disease, although 80 years ago Leyden[22] pointed out that syringomyelia in the adult is a "rest" of a congenital hydromyelia that "cuts itself off" from the central canal.

The local manifestation of atresia in the adult is a membranous remnant of the embryonic rhombic roof which occludes the foramen of Magendie and most commonly produces a mild Arnold-Chiari malformation or a cerebellar hernia.[12] Under the steady pounding of the pulse wave of the ventricular fluid, the occluding membrane eventually may perforate; but since meanwhile the surrounding structures have become impacted in the foramen magnum, obstruction of the foramen of Magendie persists. From then on, regardless of whether the foramens of Luschka open, there will remain a partial block between the cranial and spinal subarachnoid spaces. The block is more complete during systole as the impaction is rendered more pronounced by the accompanying downward excursion of the cerebellar tonsils into the funnel-shaped foramen magnum. In response to the friction of the pulsating structures incarcerated in the foramen magnum, a fibrous thickening of the meninges may develop at this level (pachymeningitis cervicalis).

Although in patients having syringomyelia there is no increase in intracranial pressure, there frequently is ventricular dilatation.[15] This dilatation represents hydrocephalus that became compensated during prenatal or post-

natal life, thus permitting the patient to survive to adulthood. The mild form of embryonal atresia that results in mild Arnold-Chiari malformation and eventuates in the clinical picture of syringomyelia in the adult, seldom produces recognizable symptoms or signs early in life.

The Living Pathology

Although the upper portion of the central canal connecting the fourth ventricle with the hydromyelic sac is patent, it seldom is involved in the dilatation because of the resistance offered by the interlaced decussating tracts in the medulla as well as by the impaction of the herniated structures in the foramen magnum. This absence of dilatation of the upper portion of the central canal, together with the damage to this area which occurs during the standard postmortem removal of the brain, has caused investigators to overlook the connection that exists between the fourth ventricle and the syrinx.

The force of the pulse wave of the ventricular fluid (shown by Bering[21] to consist of a steeply rising gradient and a gradual fall) causes the narrow upper portion of the central canal to act like a one-way valve, so that part of the fluid that enters it during systole remains trapped below during diastole. This hydrodynamic mechanism is aided by the fact that the spinal portion of the dural sac is distensible by virtue of its compressible epidural venous plexus, whereas, the cranial portion is not. When the obstruction of the foramen of Magendie is released at operation, the pulse wave of the ventricular fluid, previously funneled toward the central canal, is shunted into the subarachnoid space on the outer surface of the cord as nature intended. There its force tends to collapse the dilated central canal. This hydrostatic mechanism, so clearly apparent during operation on the living patient, can only be suspected on the basis of the findings at necropsy.

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PREVENTION OF OVEROXYGENATION DURING TREATMENT WITH A HEART-LUNG MACHINE IN CARDIAC OPERATIONS

Use of Clark Polarograph for Regulating Oxygen Tension

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MANY improvements have been made on oxygenators of heart-lung machines to assure sufficient oxygenation of blood, but little attention has been given to the problem of avoiding *over*oxygenation of blood. The possible dangers of overoxygenation, and its prevention by means of a polarograph are discussed in this paper.

Overoxygenation of blood is likely to occur under the following conditions: (1) during recirculation of blood through an oxygenator prior to connection of a patient to the machine; (2) during partial cardiac bypass with a small flow rate, which is a routine procedure at the beginning and at the end of many perfusions; and (3) during perfusion of a small patient with an oxygenator of large capacity.

Our use of the polarograph in the control of oxygenation was based on our impression that some sudden and unexpected fatalities that occurred 10 to 12 hours after perfusion might be due to overoxygenation of the patients' blood during treatment with heart-lung machines. Kirklin[1] has reported a similar impression.

Effect of Oxygen Excess

The data on toxicity of oxygen are scarce. Ninety to 95 per cent of oxygen at atmospheric pressure can be breathed by man for short periods without ill effects; however, Pichotka and Kühn[2,3] found that in guinea pigs and rabbits degenerative changes of the liver occur after exposure to 90 per cent of oxygen for eight hours every day for 90 days, and that death eventuates after continuous exposure of from 65 to 75 hours. Necropsy of these animals showed severe damage of the lungs, consisting of thickening of alveolar wall with desquamation of epithelium, pulmonary edema, and pleural exudation; dilatation of the heart; necrosis of the myocardium; and congestion of the liver and the kidney.**

This study was supported by a grant to Doctor Kolff from the Cleveland Area Heart Society. Mr. Frederick Olmsted gave valuable advice.

*Fellow in the Division of Research.

***Retrolental fibroplasia in premature children maintained in an oxygen tent also is attributed to high oxygen tension. (Arnall Patz in Report of 16th M. and R. Pediatric Research Conference on Retrolental Fibroplasia, Role of Oxygen, 1955.)*

Exposure of animals to oxygen at 3 or 3.5 atmospheres of pressure is more harmful than exposure at atmospheric pressure. Under such high oxygen pressure all hemoglobin in venous blood exists as oxyhemoglobin and thus the hemoglobin, the most important mechanism for carrying CO_2, is eliminated.[4] This results in the retention of CO_2 in peripheral tissues. Under such circumstances animals usually die within a few hours from tissue acidosis. Suppression of pyruvate oxidase and impairment of oxidative metabolism of carbohydrate in the central nervous system also is attributed to the acute oxygen intoxication.[5] According to the results of experiments on isolated slices of brain,[6] high oxygen pressure causes poisoning of many of the enzymes concerned with oxidative processes, thus paradoxically inducing cerebral anoxia. Dickens[7] recently collected available data and arguments to show the toxicity of high oxygen tension on nervous tissue.

If a human being is exposed to a high pressure of oxygen, manifestations of oxygen intoxication rapidly appear. A common early symptom is twitching of the lips or in the arms; other symptoms are dizziness, nausea, vomiting, irregular respiration, confusion, and in some instances convulsions and unconsciousness.[8]

Though oxygenation of blood by most heart-lung machines takes place under atmospheric pressure, the partial pressure of oxygen in the oxygenators can approach 700 mm. Hg if the blood is oxygenated with oxygen plus 1.5 per cent of CO_2 at a barometric pressure of 760 mm. Hg and at a temperature of 37 degrees Centigrade (the water vapor pressure is 47 mm. Hg and 1.5 per cent of CO_2 would account for 11 mm. Hg.). Recently Penido, Swan, and Kirklin[9] warned of the possibility of overoxygenation of blood with a bubble oxygenator, particularly when there is a temperature gradient between the oxygenator and the patient's body. Clark's oxygen dispersion oxygenator circumvents this problem.[10] The solubility of oxygen in blood decreases with a rise in temperature.[11] If, for example, the temperature of the blood of 30 degrees Centigrade in the oxygenator increases to 37 degrees in the patient's body, the pO_2 would increase from 700 to about 800 mm. Hg.[12] It is conceivable that such increased oxygen tension harms the patient, especially when the blood flow is large in relation to the physiologic demands of the patient, or when cavitation occurs in areas of high velocity flow.

Prevention of Overoxygenation of Blood in Oxygenators

Clark, Wolf, Granger, and Taylor[13] have pointed out that there is a proportional relationship between polarographic reading and oxygen tension. On the other hand, oxygen content increases only slightly after the hemoglobin has been fully oxygenated. As seen in Figure 1 the oxygen tension rises from 140 to 690 mm. Hg while the oxygen content increases only 1.4 volumes per cent, most of which represents physically dissolved oxygen. At an oxygen tension of more than 400 mm. Hg, no further increase in oxygen saturation of hemoglobin occurs.[14] Thus, measurement of oxygen tension by a polarograph provides

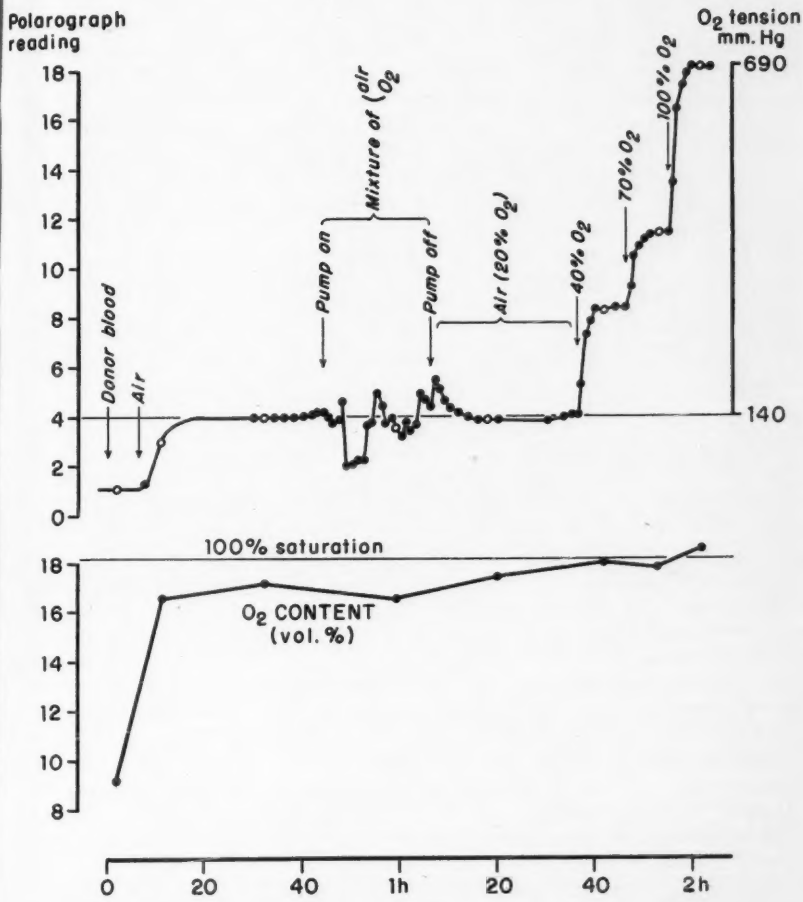

Fig. 1. Record of polarographic reading actually is a reading of the galvanometer scale in one case. Before the perfusion the polarographic reading gradually increases as the donor blood is oxygenated by 98.5 per cent of air and 1.5 per cent of CO_2 and reaches a stabilized level that is used as a base line. During the bypass period (from "pump on" to "pump off"), the polarographic reading is maintained near the base line by sending suitable gas mixtures to the oxygenator. The blood in the oxygenator was overoxygenated purposely after the perfusion had been completed. The polarographic reading and oxygen content obtained from over-oxygenated blood are presented. As the oxygen tension of blood rises, the polarographic reading increases proportionally while the oxygen content increases only slightly. All gas mixtures sent to the oxygenator contained 1.5 per cent of CO_2 to maintain a constant pH.

a better estimate of high oxygen values than does measurement of oxygen content or oxygen saturation, and polarography is most suitable for the study and prevention of overoxygenation.

The polarograph* we use is that described by Clark and associates.[15] Briefly, the polarographic determination of oxygen depends upon the reduction of oxygen by hydrogen ions at a cathode maintained near 0.6 volt, according to the equation: $O_2 + 4H^+ + 4e = 2H_2O$. The tiny current that flows as a result of this electrolysis is measured through a suitable circuit by a galvanometer. The amount of current flowing is directly proportional to the oxygen tension in the electrolyte solution, if other variables are controlled or corrected. The electrode of the Clark polarograph consists of a silver chloride anode and a platinum cathode bathed in saturated potassium chloride solution and covered by a polyethylene membrane. The polyethylene membrane readily permits the passage of oxygen but prevents the exchange of electrolytes between blood and potassium chloride solution. As the polarograph utilizes oxygen the blood must be vigorously stirred. Changes in the temperature of blood influence the readings.

Technic

For this study the Björk rotating disc oxygenator modified by Kay and associates**[16] was used. The electrode of the Clark polarograph (Fig. 2) was inserted in the oxygenator on the arterial or outflow end. Two gas tanks were prepared, one containing 98.5 per cent of air and 1.5 per cent of CO_2, and the other containing 98.5 per cent of oxygen and 1.5 per cent of CO_2. This amount of CO_2 maintained the pH of the blood within approximately normal limits.[17] Prior to connecting the machine to the patient the donor blood was oxygenated in the oxygenator by the air-CO_2 mixture until the reading of the galvanometer connected with the polarograph became stabilized at a certain level. The oxygen tension of the blood was then considered to be about 140 mm. Hg [(760-47) x 98.5% x 20%], which is higher than that of normal arterial blood (90 to 100 mm. Hg), and oxygenation of the hemoglobin must be close to 100 per cent.

The polarographic reading obtained from this air-oxygenated blood was then used as a base line. During cardiac bypass adequate amounts of oxygen (with CO_2) from the second tank were mixed with the air going into the oxygenator so that the polarographic reading was roughly maintained at this base line. The speed of the rotating discs also was adjusted. By these procedures both overoxygenation and underoxygenation of the blood were avoided.

Made by Yellow Springs Instrument Company, Inc., Yellow Springs, Ohio.

**Kay and Cross oxygenator made by Pemco, Incorporated, 5663 Brecksville Road, Cleveland, Ohio.*

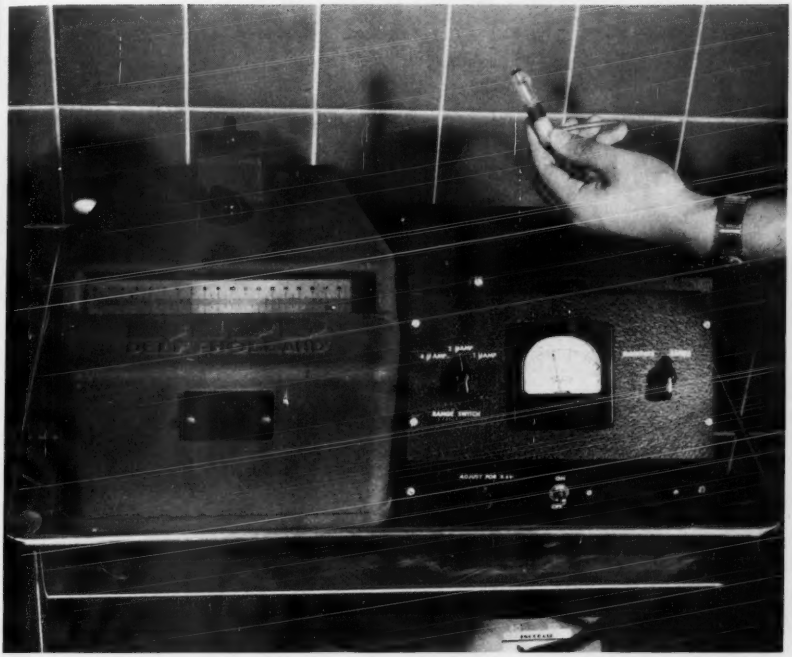

Fig. 2. Photograph of the Clark electrode with its polarograph and galvanometer. The electrode is covered with polyethylene membrane and is fixed in the oxygenator near the outflow end.

Results

The Clark polarograph connected to a heart-lung machine has been used and the data in 20 consecutive patients have been analyzed.* The patients' ages ranged from 20 months to 53 years, and the weights varied from 9.5 to 77.3 kg. The rates of perfusion ranged from 40 to 120 ml. per kilogram per minute, and the duration of the perfusion from 17 to 67 minutes. Cardiac arrest by potassium citrate, lasting from 5 to 29 minutes, was used in all cases. Nineteen of these 20 patients recovered; the one death was unrelated to problems of oxygenation as here discussed.

The polarographic readings during bypass show some fluctuation but generally they remain near the base line as seen in Figure 3. In two cases,

L. K. Groves, M.D., and W. V. Martinez, M.D., of the Department of Thoracic Surgery, co-operated in this work.

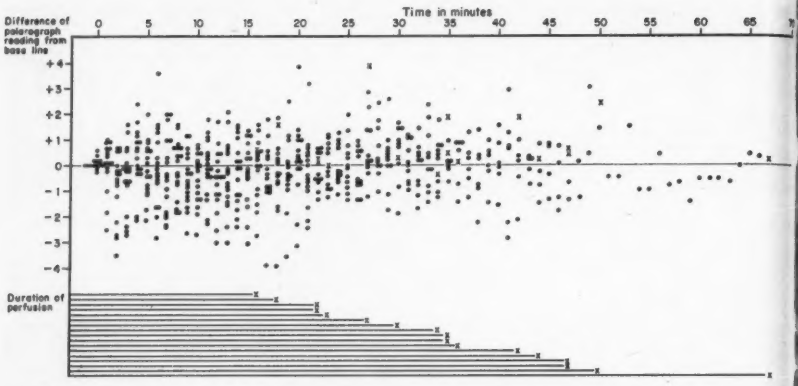

Fig. 3. The differences between the polarographic readings during perfusion and the base-line readings are plotted for 17 patients. (Polarographic readings in three patients are not included. Two will be presented separately and in the third the data were not complete. The base line indicated by **O** corresponds to about 140 mm. Hg of oxygen tension. **X** represents the end of perfusion. In most cases oxygen tension of the blood in the oxygenator was maintained near the base line throughout the perfusion. The actual reading of the galvanometer for the base line in our setup ranged from 4.0 to 6.1.

Patients 105 and 111, abnormal polarographic readings were recorded. Patient 105 (Fig. 4) weighed 75 kg. and was perfused with a flow rate of from 50 to 60 ml. per kilogram per minute. The patient's body weight proved to be above the upper limit of the oxygenating capacity of a 20-inch Kay and Cross oxygenator. The readings indicated that the blood was not sufficiently oxygenated. However, as the base line of 6.0 corresponds to an oxygen tension of 140 mm. Hg, a polarographic reading of 3.0 indicates an oxygen tension of about 70 mm. Hg. According to the oxygen-dissociation curve of blood,[18] this corresponds to 90 per cent of oxygen saturation of hemoglobin. In Patient 111 (Fig. 5) an example occurred of an increased base line. Usually our base line lies between 4 and 6 but in this case the reading of the base line was 9.2. This may be due to a change in permeability or thickness of the polyethylene membrane or other variables. At the end of perfusion, oxygenation with air (+1.5 per cent CO_2) returned the readings to the same base line. Although in this case the condition was not ideal, the polarograph still gave a fair estimate of oxygenation. In both Patients 105 and 111 the postoperative course was uneventful.

Summary

Overoxygenation of blood in heart-lung machines may in the past have led to sudden death in the postoperative phase.

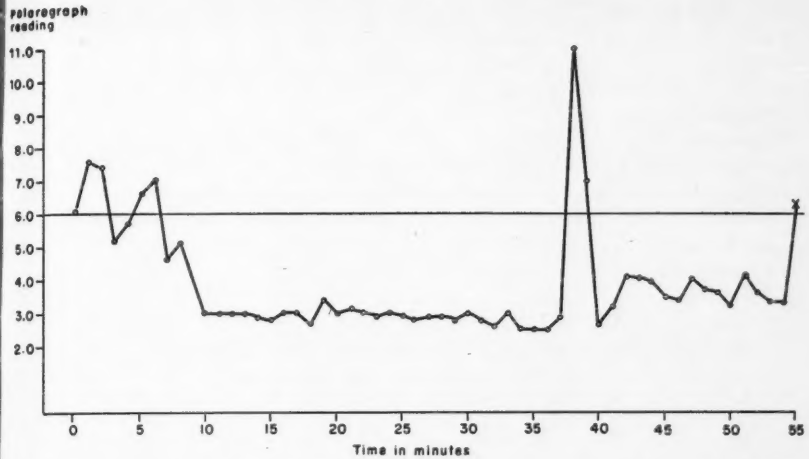

Fig. 4. (Case 105.) The low polarographic readings show insufficient oxygenation which probably resulted from the great body weight of the patient in relation to the capacity of the oxygenator. However, the reading of 3.0 indicates about 70 mm. Hg of oxygen tension, which still corresponds to 90 per cent of oxygen saturation of hemoglobin. The high peak of the polarographic readings was caused by a temporarily reduced flow through the oxygenator.

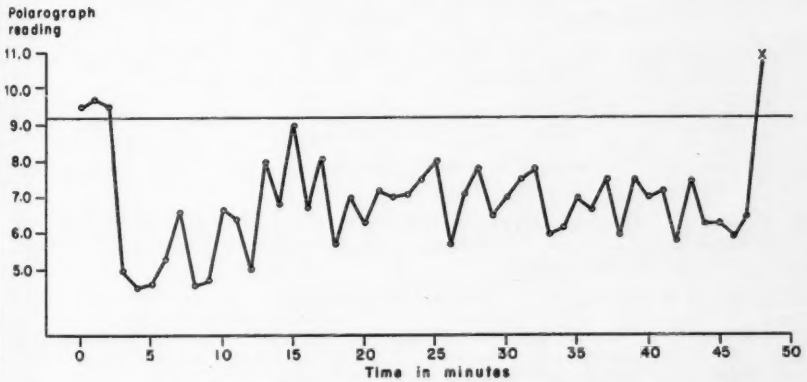

Fig. 5. (Case 111.) An unusually high base-line reading was obtained: the reading of 5.0 is considered to correspond to more than 70 mm. Hg of oxygen tension.

Overoxygenation can occur under the following conditions: (1) during recirculation of blood through an oxygenator prior to connection of a patient to the machine; (2) during partial cardiac bypass with a small flow rate, which is a routine procedure at the beginning and at the end of many perfusions; and

(3) during perfusion of a small patient with an oxygenator of large capacity. A Clark polarograph was connected to a heart-lung machine to avoid overoxygenation of blood. The polarograph measures oxygen tension, which provides a better estimate of high oxygen values than does measurement of oxygen content or of oxygen saturation.

Results are reported in a series of 20 consecutive patients in whom oxygen tension of blood had been regulated according to polarographic readings during treatment with a heart-lung machine. There has been one postoperative death in these 20 consecutive cases and this was not related to the problem here discussed.

Acknowledgment

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PROSTHETIC REPLACEMENT OF THE HEMISECTED MANDIBLE

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RECONSTRUCTION of one half of the mandible including its condyle is a formidable surgical problem. Ideally it should be possible to graft a piece of bone that will unite with the remaining bone segment at one end, and form an articulating condyle at the other. Unfortunately, the graft does not behave in this ideal fashion, and for practical purposes is unsatisfactory. Because of this difficulty, many surgeons believe that the mandibular fragment is better left unsupported after hemisection. They consider the inevitable collapse of the pharynx neither disabling nor dangerous if a tracheostomy is maintained for a sufficiently long period, and they argue that the secondary deformity associated with abnormal mobility of the jaw fragment is inconsequential.

Despite these arguments, there is no question that pharyngeal collapse, even if it does not increase the chance of mortality, is most unpleasant to the patient, particularly if it is prolonged. Furthermore, while a majority of patients will tolerate a jaw segment that, when chewed with, wanders across much of the lower half of the face, they are most grateful if reasonably normal function can be established. For these reasons, the remaining portion of mandible should be supported in its normal position at the time of resection, if such support is possible.

Probably every surgeon who has removed a number of jaws has attempted such reconstruction. Because of a long history of bone-graft failures, most of these attempts have been carried out with prosthetic devices of inert metal or plastic, shaped to resemble the resected specimen. These efforts generally have met with failure, with ultimate extrusion of the large foreign body. The series of cases reported by Healy and associates[1] using acrylic prostheses is illustrative: of their eight implants, most were extruded, and only one stayed in place for as long as one year. Believing the size of the prosthesis to be the most significant factor in this lack of success, Byars[2] replaced the hemisected jaw with a small Steinman pin, the condylar end of which terminated in an olive-shaped piece of stainless steel drilled through in numerous places to permit its transfixion by granulation and scar tissue. His results have been consistently superior to those previously reported. We agree heartily with Byars' concept, and have employed a similar prosthesis modified to minimize its mass and to simplify its construction.

Technic

The prosthesis we employ is shown in Figure 1. It consists of a Steinman pin that in cross section measures approximately $\frac{1}{16}$ inch. Its condylar portion is a $\frac{9}{32}$ inch stainless-steel ball bearing, drilled and driven into position on the pin. If the joint is tight, soldering or brazing is unnecessary. It has not been found necessary to drill additional holes through this ball for fixation by fibrous tissue. At operation the pin is bent to the desired angle (Fig. 1).

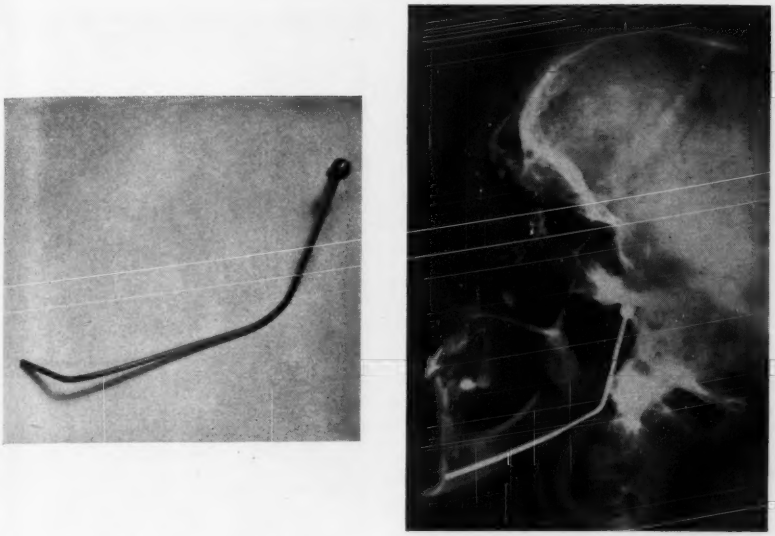

Fig. 1. (Left) Photograph of the prosthetic device bent to shape for implantation. (Right) Postoperative roentgenogram showing the prosthesis in place.

After hemisection of the jaw, a pin-sized hole at least ¾ inch long is drilled into the cut end of the remaining jaw fragment. With any available teeth in occlusion, this hole should accept the pin without torsion or displacement of the prosthetic condyle out of the fossa. The pin is angled so that its course from condyle to bone is just in contact with the soft tissue medial to it. This makes it possible to close the soft tissue over the pin without tension. Such closure prevents contact of the pin with the skin flap anywhere along its course, thus minimizing the possibility of subsequent erosion through the skin. Because of its light weight and inconsequential mass the prosthetic condyle remains in the fossa without additional support. Except for the skin approximation, 4-0 chromic catgut sutures are employed throughout. A mild pressure dressing is applied at the close of the procedure. A Levin tube and a prophylactic tracheostomy are essential only during the immediately postoperative period and are discontinued as soon as possible. Minimal interdental wiring with rubber band support is applied at the time of operation if teeth are available.

Results

During the past four years, we have used this type of prosthesis on five patients. Two of these implants are currently functional after four (Fig. 2) and

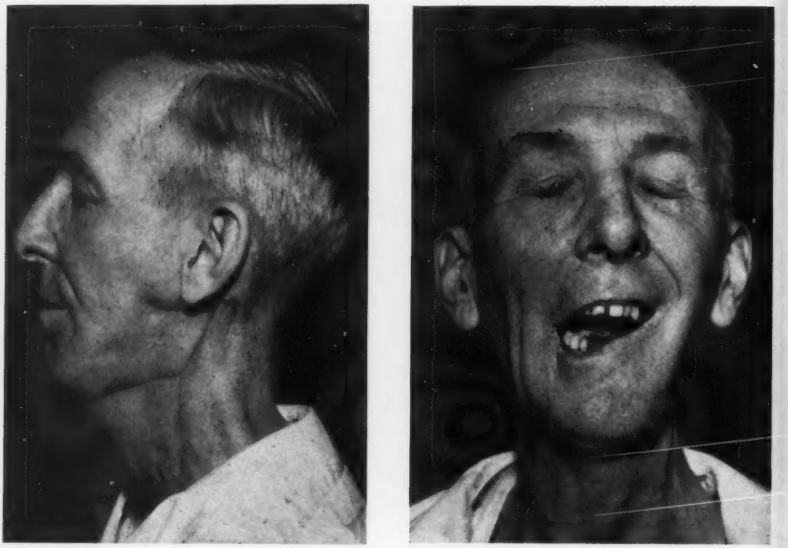

Fig. 2. (Left) Lateral view of patient with prosthesis in place for four years, showing minimal deformity. (Right) The patient opens his mouth easily, adequately, and in the normal plane of mandibular motion.

two years respectively. A third was tolerated well but was removed at the time of subsequent surgery for a recurrence of tumor approximately one year after insertion. The remaining two were not tolerated because of local infection and had to be removed in the early postoperative period; in neither of these was erosion of skin a factor.

Summary

When one half of the mandible is resected, the remaining jaw fragment should be supported by means of an articulating prosthesis. A stainless-steel implant of minimal size has been effective for this purpose.

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OBSERVATIONS CONCERNING RENAL CIRCULATION

Angiography of Kidneys Removed from Hypertensive or Normotensive Patients

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SINCE aberrations in the renal circulation in experimental animals can be demonstrated by angiography,[1-5] the present study was undertaken for two purposes: (1) to determine whether there are roentgenographically detectable alterations in the arterial system of kidneys removed from hypertensive patients; and (2) if such alterations are present, to determine whether they can be recognized by clinical renal angiography.

Material and Methods

Twenty-nine excised kidneys were selected from necropsy or from surgical specimens. Sixteen of the kidneys were from hypertensive and 13 from normotensive patients.

Angiography of excised kidneys. A barium-latex suspension having a particle size of approximately 30 microns was injected through a cannula into the main renal artery of each kidney. This particle size prevented the passage of the material through the capillaries into the venous system. To obtain good renal filling, the suspension was injected by manometric control under a pressure greater than the patient's usual diastolic pressure by from 10 to 20 mm. Hg. In a few instances higher pressures were used, but there was no apparent increase in the vascular filling. The cannula was removed and the vessels were ligated. Roentgenograms of the kidney were then made using a cardboard technic with a 40-inch focal-film distance. The technical factors were 50 milliamperes, 35 kilovolts, and 1½ seconds.

Findings

The pathologic diagnoses of the conditions of the 29 kidneys are listed in Table 1.

Arterial system in normal kidneys from normotensive patients. The roentgenograms of the three normal kidneys (Group 1, Table 1) were notable because of three

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features (Fig. 1): (1) uniformity of luminal size in the large and medium-sized vessels and the interlobar and arcuate arteries; (2) good filling of the interlobular arteries (cortical arterioles) that usually arise in a fanlike pattern from the arcuate arteries and supply the cortex of the kidney; and (3) uniform distribution of those terminal vessels that extend almost to the periphery of the renal parenchyma (Diagram 1, Fig. 1).

Table 1.—*Pathologic diagnoses of conditions of kidneys excised from 29 hypertensive or normotensive patients*

Group	Pathologic diagnosis	Number of patients, total	hypertensive	normotensive
1	Normal	3	0	3
2	Vascular disease	10		
	Arteriolar nephrosclerosis 8		8	0
	Polyarteritis nodosa 1		0	1
	Aneurysm of renal artery 1		0	1
3	Disease of glomerulus	6		
	Glomerulonephritis 4		4	0
	Intercapillary glomerulosclerosis . . . 1		1	0
	Lupus erythematosus 1		1	0
4	Infectious renal disease	6		
	Pyelonephritis 3		0	3
	Acute necrotizing pyelonephritis 1		0	1
	Acute suppurative nephritis 1		1	0
	Hydronephrosis and pyelitis 1		0	1
5	Anoxia	2		
	Lower nephron nephrosis 2		0	2
6	Other disease	2		
	Congenital hypoplasia 1		1	0
	Renal cyst 1		0	1
	Total	29	16	13

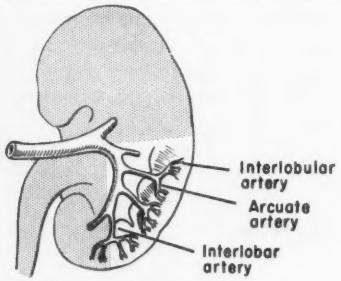

Diagram 1. Wedge section of the normal kidney showing the anatomy of the renal artery and its branches.

Arterial system in pathologic kidneys from normotensive patients. Two of the 10 patients having vascular disease (Group 2, Table 1) were normotensive. One had polyarteritis nodosa and the other had a calcified congenital aneurysm of

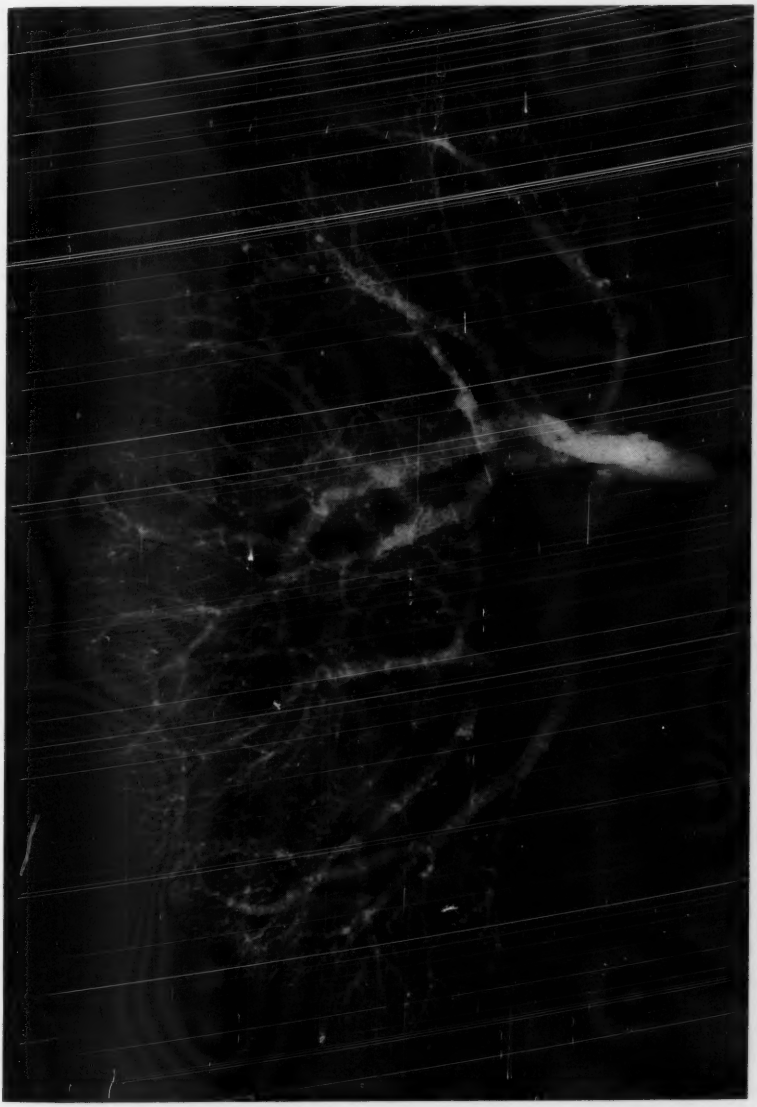

Fig. 1. Angiogram of kidney at necropsy, from a 28-year-old normotensive patient who died after craniotomy. The arterial pattern is normal: the cortical arterioles (interlobular arteries) are well filled and extend to the periphery of the cortex; the interlobar arteries are uniform in distribution and luminal size.

the renal artery. The arterial pattern in the kidney from the patient with polyarteritis nodosa was notable because it was the only one in this series which indicated a pathognomonic abnormality of the small vessels (Fig. 2). The filling of the arterial tree was complete, but numerous small saclike outpouchings extended from the vessels. These outpouchings, which represented micro-

Fig. 2. Angiogram of kidney at necropsy, from a 57-year-old normotensive patient who had polyarteritis nodosa and who died from perforation of the ileum as a result of infarction. There is normal filling of the cortical arterioles. The significant finding is the multiple, small, saclike out-pouchings of the medium- and small-sized arteries which represent aneurysms.

aneurysms, had resulted from focal necrosis of the arterial walls. Although these aneurysmal abnormalities of polyarteritis nodosa long have been recognized histopathologically, we believe that this is the first report of their recognition on roentgenograms. The renal roentgenogram of the patient with the aneurysm of the main renal artery showed a fairly normal arterial pattern distal to the aneurysm (Fig. 3).

Five of the six patients having infectious renal disease (Group 4, Table 1) were normotensive. Roentgenograms of the three kidneys from patients having pyelonephritis showed no striking abnormality and showed fairly normal filling of the terminal vessels; the diagnosis of pyelonephritis was incidental and none of the patients died of renal disease. The roentgenogram of the kidney from the patient having acute necrotizing pyelonephritis and that from the patient having hydronephrosis and pyelitis also gave no indication of arterial abnormality in so far as the injection pattern was concerned.

Both of the patients having lower nephron nephrosis (Group 5, Table 1) were normotensive, and the renal angiograms of both patients showed fairly normal filling of the terminal vessels (Fig. 4).

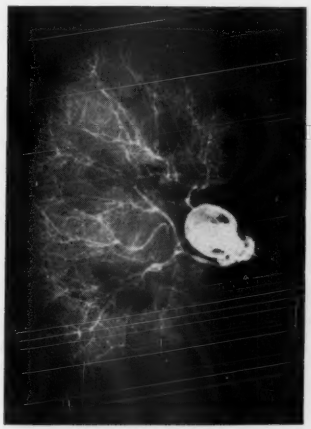

Fig. 3. Angiogram of excised kidney from a 57-year-old patient having aneurysm of the main renal artery. Preoperatively, the blood pressure was 175/80 mm. Hg and the blood-urea nitrogen content was 21 mg. per 100 ml. The aneurysm is well outlined by the injected material, and the cortical arterioles are well filled. There was no pathologic evidence of nephrosclerosis.

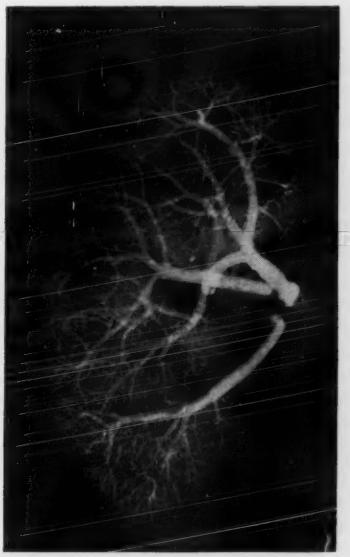

Fig. 4. Angiogram of kidney at necropsy, from a 66-year-old patient having early lower nephron nephrosis following pneumonectomy for carcinoma of the lung. The blood pressure had been 130/68 mm. Hg and the blood-urea nitrogen content 21 mg. per 100 ml. There is a normal arterial pattern similar to that shown in Figure 1.

One of the two patients in Group 6 (Table 1) was normotensive. The pathologic diagnosis was a renal cyst. A roentgenogram of the kidney from this patient showed a normal arterial pattern.

Arterial system in pathologic kidneys from hypertensive patients. Patients were considered hypertensive who had elevation of systolic and diastolic blood pressure and who showed pathologically demonstrable effects of hypertension. In contrast to the normal arterial pattern observed roentgenographically in the kidneys from all 13 normotensive patients, an abnormal pattern—diminution or absence of filling of the interlobular arteries—was observed roentgenographically in the kidneys from all 16 hypertensive patients.

Eight of the 10 patients having vascular disease (Group 2, Table 1) were hypertensive. The pathologic diagnosis of the condition of the kidney from each of these eight patients was arteriolar nephrosclerosis. In all eight, the renal angiograms after injection of the barium-latex suspension showed diminution or absence of filling of the interlobular arteries (cortical arterioles), indicating the pathologic abnormality of intimal thickening and resultant reduction in luminal size (Fig. 5).

All six of the patients having glomerular disease (Group 3, Table 1) were hypertensive. The arterial pattern of the kidneys from the four patients having chronic glomerulonephritis was similar to that of the patients having arteriolar nephrosclerosis. The striking roentgen feature was the diminution or absence of filling of the interlobular arteries (Fig. 6). The more advanced changes of glomerulonephritis also were reflected in the arterial pattern by changes in the interlobar arteries (Fig. 7). These changes, similar to those observed in the later stages of nephrosclerosis, consisted of irregularity of luminal size. The histopathologic changes in the interlobar arteries were those of nephrosclerosis, which usually is associated with advanced glomerulonephritis. The roentgen arterial patterns of the kidneys from the patient having intercapillary glomerulosclerosis and from the patient having lupus erythematosus showed diminution or absence of filling of the interlobular arteries.

One of the six patients having infectious renal disease (Group 4, Table 1) was hypertensive. The pathologic diagnosis of the kidney from that patient was acute suppurative nephritis. The roentgen arterial pattern of this kidney (Fig. 8) was essentially the same as that in the nephrosclerotic and glomerulonephritic kidneys, and involved diminution or absence of filling of the interlobular arteries.

One patient in Group 6 (Table 1) was hypertensive. The pathologic diagnosis was congenital hypoplasia. The term "congenital hypoplasia" refers to a structurally normal but small kidney and is distinct from the atrophic pyelonephritic kidney. The roentgenogram of the kidney from this patient showed a small parenchymal mass and an arterial pattern similar to that seen in the kidneys from other hypertensive patients. Arteriolar nephrosclerosis was present.

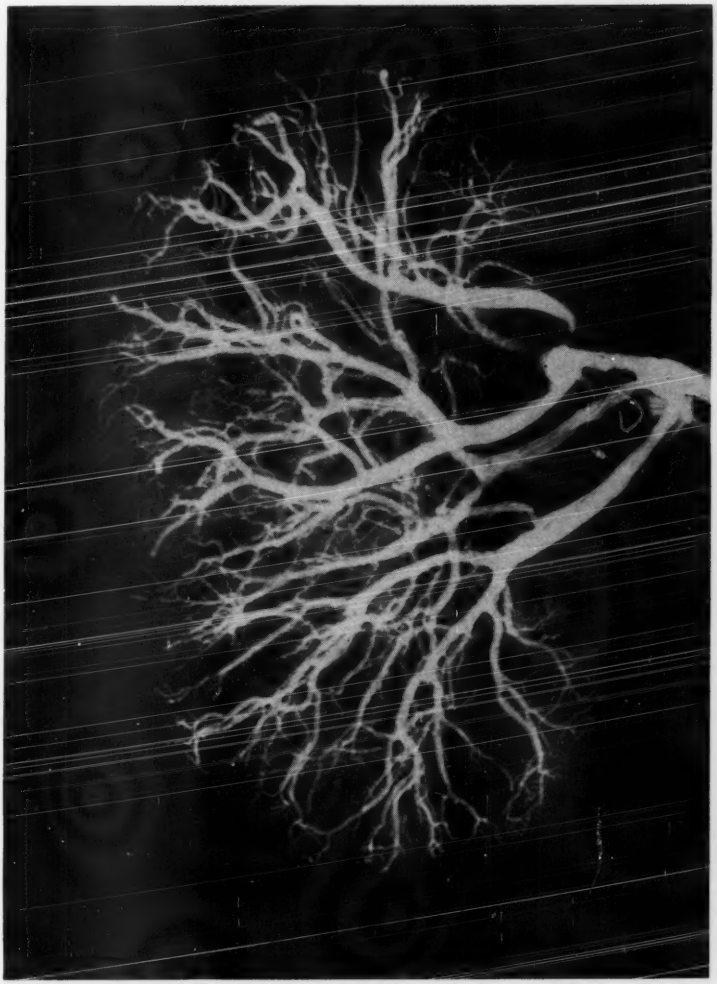

Fig. 5. Angiogram of kidney at necropsy, from a 53-year-old patient who had arteriolar nephrosclerosis and who died of cerebral hemorrhage. The blood pressure had been 266/152 mm. Hg and the blood-urea nitrogen content 102 mg. per 100 ml. There is pronounced diminution in the filling of the cortical arterioles (compare with Figure 1). Pathologically, there were marked intimal thickening and reduction in the luminal size of small arteries and arterioles.

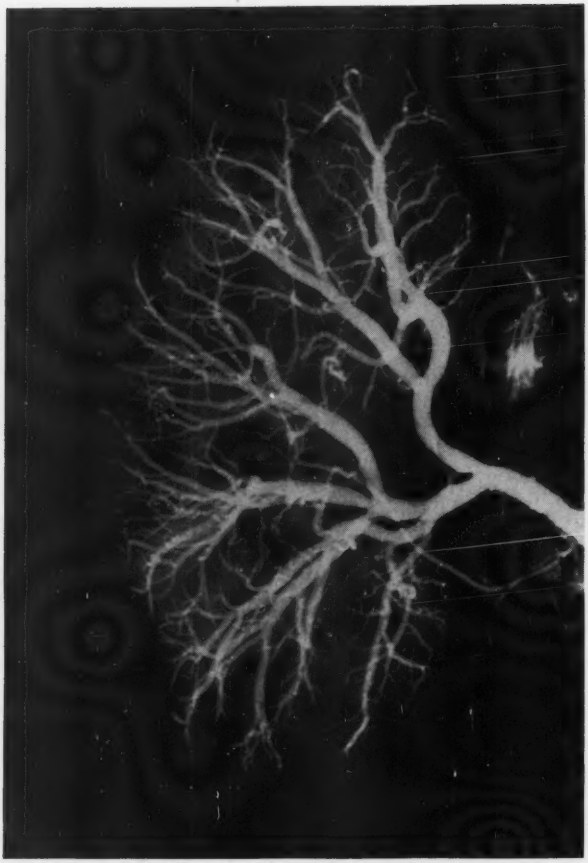

Fig. 6. Angiogram of kidney at necropsy, from a 60-year-old patient who died of chronic glomerulonephritis. The blood pressure had been 210/100 mm. Hg and the blood-urea nitrogen content 158 mg. per 100 ml. The arterial pattern is similar to that shown in Figure 5, and correlated with the pathologic findings of intimal thickening and luminal narrowing.

Comment

The roentgenograms of the kidneys from the 16 hypertensive patients showed a characteristic abnormality after barium-latex suspension had been injected into the kidneys—diminution or absence of filling of the terminal arteries. None of the roentgenograms of the 13 normotensive patients showed that abnormality. In the renal angiograms of the patients having advanced vascular disease associated with hypertension, irregularities also were observed

in the luminal caliber of the medium-sized vessels. The pathologic findings confirmed these angiographically observed abnormalities and indicated definite reduction in luminal size of the cortical arterioles. This pattern of arteriolar nonfilling in the cortical region of the kidneys from patients with hypertension is strikingly similar to the arteriolar pattern observed in animals having renal cortical ischemia under various experimental conditions including induced hypertension. In this study, the angiographic findings in kidneys from hypertensive patients indicate a reduction in luminal size and, by inference, the presence of cortical ischemia.

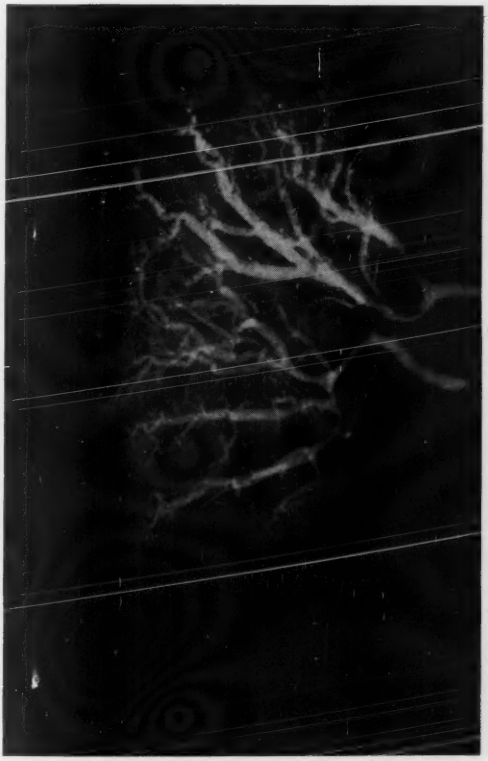

Fig. 7. Angiogram of kidney at necropsy, from a 60-year-old patient who died of advanced chronic glomerulonephritis. The blood pressure had been 170/90 mm. Hg and the blood-urea nitrogen content 192 mg. per 100 ml. There are extreme variation in luminal size of the medium-sized vessels and scant filling of the cortical arterioles which are irregular in size and distribution. (One branch of the renal artery was not injected.) The vessels are similar to those shown in Figure 6, and in addition there were arteriosclerotic changes in the large vessels.

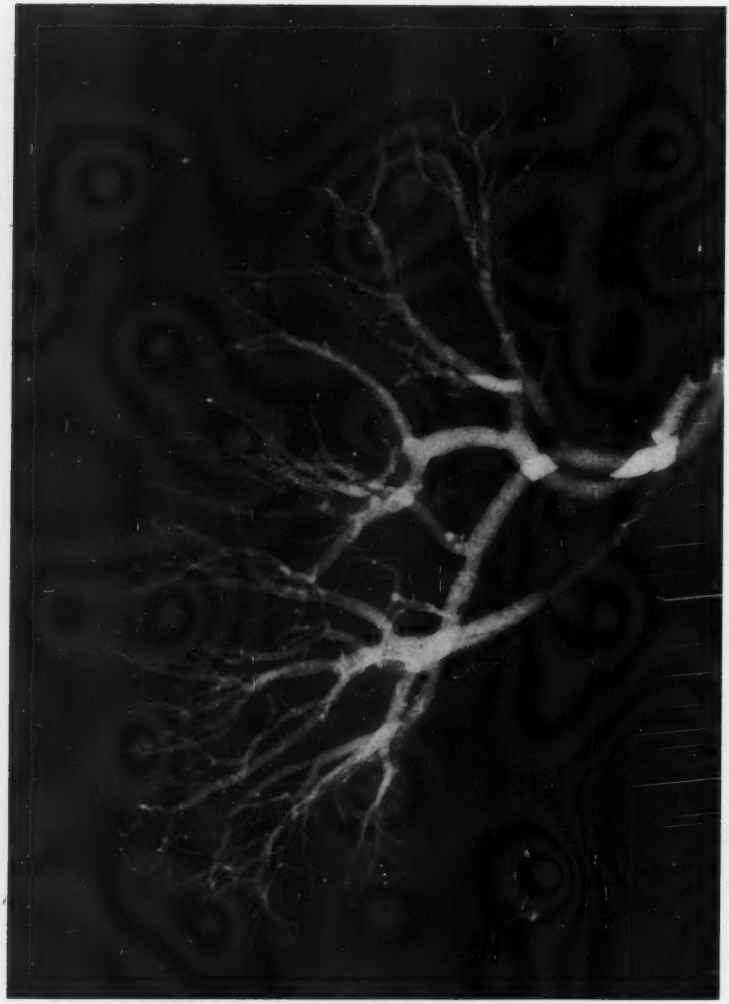

Fig. 8. Angiogram of a kidney at necropsy, from a 50-year-old patient having acute suppurative nephritis. The blood pressure had been 240/140 mm. Hg and the blood-urea nitrogen content 28 mg. per 100 ml. There is absence of filling of the cortical arterioles. Pathologic changes of arteriolar nephrosclerosis with luminal narrowing were superimposed on the suppurative nephritis.

The arterial changes in these kidneys are not always of sufficient magnitude to be demonstrable by renal aortography. However, with modification in technic, particularly by serial angiography it is sometimes possible to obtain good visualization. Thus, renal angiography may aid in the diagnosis of some of the renal diseases that manifest themselves by changes in the small renal arteries.

Summary

The arterial patterns of kidneys removed from 29 patients, 16 of whom were hypertensive and 13 of whom were normotensive, were demonstrated roentgenographically after injection of a barium-latex suspension. The arterial patterns of all of the kidneys from hypertensive patients exhibited a characteristic abnormality—diminution or absence of filling of the interlobular arteries (cortical arterioles)—which is strikingly similar to that observed in experimental animals with renal ischemia. That abnormality was not evident in any of the renal angiograms from normotensive patients.

The arterial pattern of the kidney from a patient having polyarteritis nodosa without hypertension showed multiple saclike outpouchings that we believe may be pathognomonic of this disease.

The clinical application of these findings to present-day renal angiography is sometimes limited by the inadequate visualization of these small arteries. However, when they can be visualized, the appreciation of the arterial changes may be useful in the diagnosis of renal diseases particularly those associated with hypertension.

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LEIOMYOMA OF THE TRACHEA

Report of a Case

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TUMORS rarely occur in the trachea and most of those that do occur are malignant. We are reporting a case of an unusual benign neoplasm, a leiomyoma, of the trachea which we believe is the sixth such case to be recorded in the literature.

Moersch, Clagett, and Ellis[1] in reviewing 82 cases of tracheal neoplasm classified 35 as benign and 47 as malignant tumors. However, with the exception of one, a chondroma, the lesions that they classified as benign tumors often are considered not to be truly neoplastic: papilloma, tracheopathia osteoplastica, amyloid tumor, and xanthoma. Malignant tracheal tumors are most commonly either carcinomas indistinguishable from bronchogenic carcinomas, or "cylindromas." It has been traditional to group carcinomas of cylindromatous pattern and adenomas of carcinoid structure under the term *bronchial adenoma*. We believe that this grouping is illogical because it infers that a "cylindroma" is a benign tumor. The carcinoid bronchial adenoma is a relatively benign lesion; however, by comparison, the cylindromatous carcinoma, which tends to occur more proximally in the respiratory tract, is definitely carcinomatous and usually lethal. Cylindromatous carcinoma in the respiratory tract is rare; no cases have been seen at the Cleveland Clinic since 1948, yet in these past 10 years carcinoid bronchial adenoma has been diagnosed in 21 patients.[2]

A few benign or malignant smooth-muscle tumors of the respiratory tract have been reported.[3-5] We have found references to only five, all leiomyomas, occurring in the trachea. Dorenbusch[6] reported one case in which a leiomyoma occurred a short distance below the right vocal cord on the tracheal wall; the neoplasm was removed via a cervical approach to the trachea. Greer and Winn[7] described another case in which a leiomyoma occurred in the terminal trachea arising from the membranous portion, and obstructed both main bronchi; the tumor was removed transtracheally via a right thoracotomy. Unger[8] reported a leiomyoma of the trachea in a 15-year-old girl; the tumor was treated bronchoscopically on three occasions and, according to Greer and Winn,[7] subsequently required open thoracotomy. Two other cases were cited, but no details were given, in a review by Gilbert, Mazzarella, and Feit.[9]

Report of Case

A 56-year-old man had episodes of coughing, wheezing, and shortness of breath, symptoms that had become progressively more frequent and more severe during the four years before examination in May 1955. The symptoms initially occurred only at night but later appeared at any time. In describing his respiratory distress, the patient stated that it felt as though a ball valve allowed him to inspire but prevented his expiration. On physical examination the initial impression was that the patient had bronchial asthma; but, for thoroughness, bronchoscopic examination was performed. At that examination a polypoid tumor, movable on a broad base, was found arising from the membranous portion of the trachea approximately half way between the larynx and the carina. When observed at expiration the mass was seen to fall completely across the lumen of the trachea. The tumor was soft and could be readily passed anteriorly by the bronchoscope.

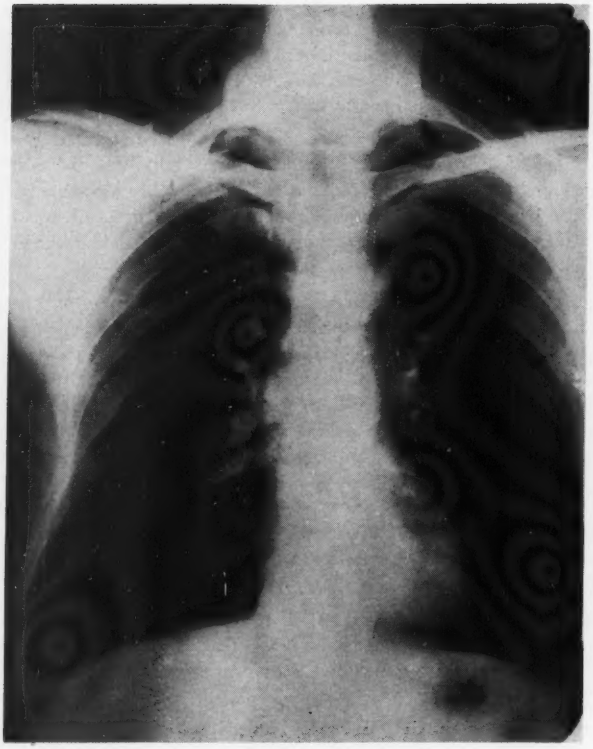
Roentgenograms of the chest demonstrated the valuable assistance that a lateral view can give. The appearance of the anterior-view roentgenogram was normal (Fig. 1A), but on the lateral-view roentgenogram, a small shadow could be seen in the superior mediastinum superimposed on the tracheal outline (Fig. 1B).

Because the tumor completely filled the trachea on expiration and because of the danger of further obstruction as a result of the trauma of bronchoscopic manipulation, the patient was hospitalized immediately and surgically treated the following day. The anesthesiologist attempted to pass a long endotracheal tube beyond the neoplasm, but some respiratory obstruction was still apparent until the chest was opened by a right thoracotomy. The tip of the endotracheal tube was then properly positioned distal to the neoplasm by direct palpation of the trachea. After the trachea was mobilized, the tumor was readily palpable immediately below the thoracic inlet. The posterior membranous trachea was incised and the tumor, including its base, was removed by sharp dissection, leaving a defect in the tracheal mucosa approximately 1 cm. in diameter. The defect was readily closed transversely with fine silk sutures.

Pathologic examination of the operative specimen revealed a smooth, glistening mass, measuring 25 by 20 by 20 mm. (Fig. 2). It seems remarkable that a mass of this size allowed sufficient airway for maintenance of life. (The cross section of the average trachea is stated to be 16.5 by 14.4 mm.[10]) The mass was composed of white tissue that was firm and rubbery.

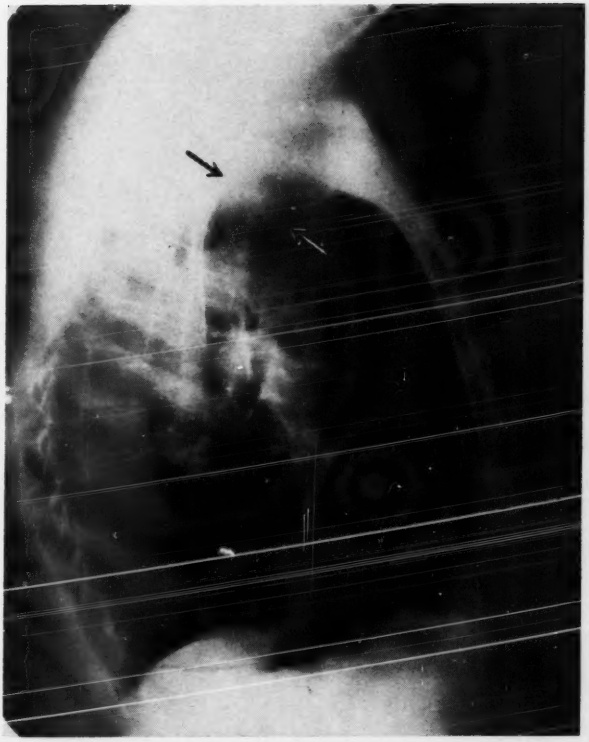
Microscopically, the tumor in large part was covered by stratified squamous epithelium. The underlying neoplasm consisted of long, slender, spindle cells arranged in anastomosing bundles (Fig. 3). The individual cell nuclei were elongated and uniform in size and shape. The nuclear chromatin was dispersed finely throughout the nucleus. No mitosis was seen. Mallory's phosphotungstic acid hematoxylin stain demonstrated relatively coarse intracellular fibrils. The fibrils occasionally terminated in small hooks (Fig. 4). Masson's trichrome stain outlined red-staining interlacing bundles of spindle cells that were separated from each other by delicate bands of green-staining collagen. No invasion of the base was apparent.

Postoperative course. The patient has been asymptomatic since operation. Findings were normal on bronchoscopic examination performed two years postoperatively. The site of the previous neoplasm could not be identified.



A

Fig. 1. Discrepant findings on roentgenograms of the chest. **A**, normal findings on the classic type of roentgenogram.



B

Fig. 1. B, lateral-view roentgenogram; arrows point to tumor mass.

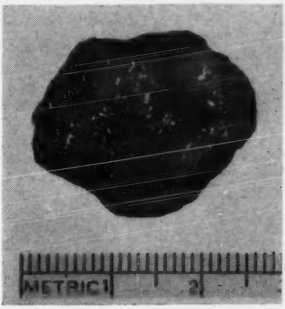


Fig. 2. Photograph of surgically removed tumor. Note its size. It was situated entirely within the tracheal lumen.

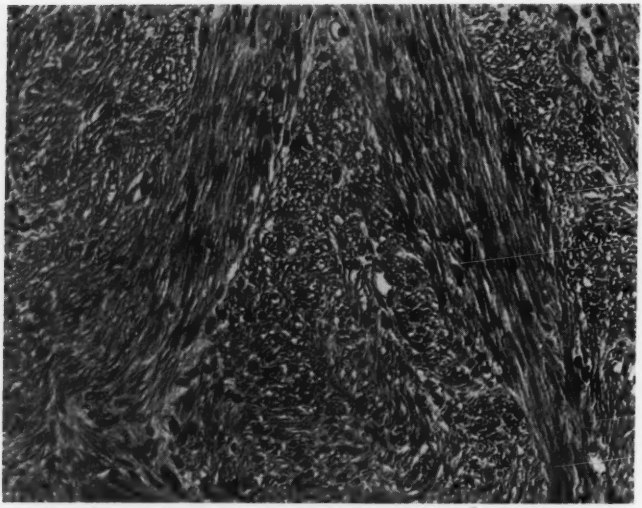


Fig. 3. Photomicrograph showing interlacing bundles of spindle cells. Hematoxylin-eosin and methylene blue; X 240.

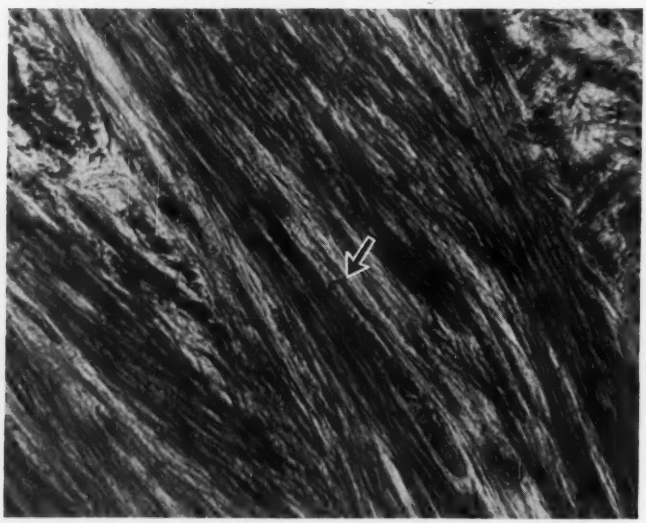


Fig. 4. Photomicrograph showing coarse intracellular fibrils. A small hook characteristic of myofibril termination is present. Mallory's phosphotungstic acid hematoxylin stain; X 700.

Summary

A case of leiomyoma of the trachea is reported because of its unusual nature and dramatic, favorable response to treatment. Because of his severe wheezing, the patient at first was considered to be asthmatic. Organic respiratory obstruction may closely mimic asthma, and frequently the two conditions may be differentiated only by endoscopic examination.

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ERRATA

In the October 1957 issue of the QUARTERLY, on page 225, lines 4, 5, and 6, the symbols for footnotes should appear as follows:

chloroquine phosphate*
hydroxychloroquine sulfate**
iproniazid+

On page 241 an acknowledgment should appear as follows:

Acknowledgment

The authors gratefully acknowledge the use of electrophoretic data supplied by Lena A. Lewis, Ph.D., Division of Research, The Cleveland Clinic Foundation.

RECENT PUBLICATIONS BY MEMBERS OF THE STAFF

ANDERSON, ROBIN: The role of the plastic surgeon in management of the severely injured patient. J.A.M.A. **165**: 1922-1923, Dec. 14, 1957.

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++Professor of Urology, University of Kansas, School of Medicine, Kansas City, Kansas.*

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THE FRANK E. BUNTS EDUCATIONAL INSTITUTE

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The auditorium capacity limits the registration to 150. Registration will follow the order in which the applications are received.

Tentative Program

Wednesday, February 12, 1958

Morning Session

8:00- 8:55	.	Registration
8:55- 9:00	.	Welcome . . . . . CHARLES L. LEEDHAM, M.D. HERBERT W. SALTER, M.D.*

Obstetrics and Gynecology

9:00- 9:20	.	Gynecologic Problems During Pregnancy . . ALBERT C. LAMMERT, M.D.
9:20- 9:40	.	Gynecologic Problems After Pregnancy . . . . . PAUL R. ZEIT, M.D.
9:40-10:00	.	Office Diagnostic Procedures in Gynecology . . JAMES S. KRIEGER, M.D.
10:00-10:30	.	Questions and Answers
10:30-11:00	.	Intermission

Pediatrics

11:00-11:20	.	The Handicapped Child . . . . . ROBERT D. MERCER, M.D.
11:20-11:40	.	The Uncommon Types of Epilepsy in Children . . . . . EDWARD M. ZUCKER, M.D.
11:40-12:00	.	Evaluating Intelligence in Children . . . . . CLARE ROBINSON, M.S.
12:00-12:30	.	Questions and Answers
12:30- 1:30	.	Luncheon—*Courtesy of Bunts Institute*

*Chairman, Education Committee, Cleveland Chapter of the American Academy of General Practice.

Afternoon Session

Therapy

1:30- 1:50 . Staples Mobilization . . . . . HAROLD E. HARRIS, M.D.
1:50- 2:10 . Symbiotic Combinations in Antibiotic Therapy . . . . . RAY A. VAN OMMEN, M.D.
2:10- 2:50 . Practical Approach to Problems of Electrolyte Balance . . . . . WILLIAM A. KELEMEN, M.D.
2:50- 3:20 . Questions and Answers
3:20- 3:50 . Intermission

Trauma

3:50- 4:10 . The Orderly Approach to the Injured Patient . . . . . STANLEY O. HOERR, M.D.
4:10- 4:30 . Management of Ocular Injuries . . . . . ROSCOE J. KENNEDY, M.D.
4:30- 4:50 . Immediate Management of Chest Injuries . . . DONALD B. EFFLER, M.D.
4:50- 5:45 . Questions and Answers
6:00- . Reception—Wade Park Manor

Tentative Program

Thursday, February 13, 1958

Morning Session

Old Timers' Session

9:00- 9:20 . Psychoneurotic States . . . . . LOUIS J. KARNOSH, M.D.
9:20- 9:40 . Common Geriatric Dermatoses . . . . . EARL W. NETHERTON, M.D.
9:40-10:00 . Functional Mechanism of Pain . . . . . CARL J. WIGGERS, M.D.
10:00-10:20 . Changing Concepts of Renal Disease . . . . . WILLIAM J. ENGEL, M.D.
10:20-10:50 . Questions and Answers
10:50-11:15 . Intermission

Miscellaneous Topics

11:15-11:45 . Cine-Cardioangiography in the Diagnosis of Congenital Heart Disease . . . . . F. MASON SONES, JR., M.D.
11:45-12:00 . Obesity and Longevity . . . . . OTTO P. SCHUMACHER, M.D.
12:00-12:20 . Dermatologic Lesions of the Anal Area . . . KENNETH H. BURDICK, M.D.
12:20-12:40 . Common Diagnoses in Office Proctology . . . . . RUPERT B. TURNBULL, JR., M.D.
12:40- 1:10 . Questions and Answers

* * *

2:10 p.m. . Tour of the Cleveland Clinic Hospital and the Cleveland Clinic

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